Please amend the following claim:

5. (THREE TIMES AMENDED) An isolated or purified polynucleotide comprising SEQ ID NO:1 or its complementary strand.

12. (THREE TIMES AMENDED) A diagnostic device comprising the nucleotide sequence of claim 5.

14. (THREE TIMES AMENDED) A pharmaceutical composition comprising a pharmaceutically acceptable carrier and the nucleotide sequence of claim 5.

REMARKS

Claim amendments were made to more clearly claim the invention. No new matter has been added. Changes to the claims can be seen on a separate page following the signature page entitle VERSION WITH MARKINGS TO SHOW CHANGES MADE. Deletions are in **[bold and brackets]** and insertions are underlined.

Rejection Under 35 U.S.C. § 112, First Paragraph

Claims 5-9, 12, 14, and 16 were rejected under 35 U.S.C. § 112, first paragraph, for lack of enablement. The Examiner stated that the specification enables polynucleotides of SEQ ID NOS: 1. The claims have been amended to recite an isolated or purified polynucleotide comprising SEQ ID NO:1 or its complementary strand. Therefore, Applicants assert that the amended claims are enabled by the specification. Applicants respectfully request withdrawal of the claim rejection on this basis.

Claims 5-9, 12, 14, and 16 were rejected under 35 U.S.C. § 112, first paragraph, for lacking written description for the claimed invention. The amended claims recite an isolated or purified polynucleotide comprising SEQ ID NO:1 or its complementary strand. The specification as filed clearly provides written description support for SEQ ID NO:1, in particular in the Sequence Listing. The complementary strand may be easily determined by anyone of skill in the art based on SEQ ID NO:1. Therefore, Applicants assert that written description support of the presently claimed invention is provided in the application as filed. Applicants respectfully request withdrawal of the rejection on this basis.

Rejection Under 35 U.S.C. § 102

Claim 8 was rejected under 35 U.S.C. § 102(b) as being anticipated by Hillier et al. (Accession No. W00593, April 1996, PTO 892) or Hillier et al. (Accession No. N91311, April 1996, PTO 892) or Hillier et al. (Accession No. W38597, May 1996, PTO 892) or Hillier et al. (Accession No. N68467, March 1996, PTO 892) or Hillier et al. (Accession No. N42215, January 1996, PTO 892) or Hillier et al. (Accession No. H20154, July 1995, PTO 892). Claim 8 has been cancelled, thus the rejection is moot.

Claims 5-9, 12, 14, and 16 were rejected under 35 U.S.C. § 102(e) as being anticipated by US Patent No. 6,197,543. The '543 patent teaches a polynucleotide that is 99.1% identical to SEQ ID NO:1. Claim 5 has been amended to recite an isolated or purified polynucleotide comprising SEQ ID NO:1 or its complementary strand. The '543 patent does not disclose SEQ ID NO:1, therefore Applicants assert that the presently claimed invention is not anticipated by the prior art. Applicants respectfully request withdrawal of the rejection on this basis.

Rejection Under 35 U.S.C. § 103

Claims 9 and 16 were rejected under 35 U.S.C. § 103(a) as being unpatentable over Hillier et al. (Accession No. W00593, April 1996, PTO 892) or Hillier et al. (Accession No. N91311, April 1996, PTO 892) or Hillier et al. (Accession No. W38597, May 1996, PTO 892) or Hillier et al. (Accession No. N68467, March 1996, PTO 892) or Hillier et al. (Accession No. N42215, January 1996, PTO 892) or Hillier et al. (Accession No. H20154, July 1995, PTO 892) or Marra et al. (Accession No. W71344, June 1996, PTO 892), each in view of Sambrook et al.

The presently claimed invention recites a vector comprising SEQ ID NO:1 or its complementary strand and a cell transformed by the vector or comprising a partial or total genomic deletion of SEQ ID NO:1, or a homologue thereof.

Recent research has shown the significance of SEQ ID NO:1 as a potential tool in diagnostic and therapeutic applications, particularly in the treatment of lung injuries and diseases or several disorders related to oxidative stress. For example, several single nucleotide polymorphisms (SNPs) of *Homo sapiens* peroxiredoxin 5 gene (PRDX5) have been reported in public databases (GenBank/NCBI Accession Nos. XM_048280 and XM_048277). Some of these

SNPs are in the coding region of the gene. PRDX5 SNPs are relevant to the identification of susceptibilty to lung injuries and diseases and oxidative stress disorders.

Furthermore, PRDX5 recombinant protein is shown to provide protection against excitotoxic insults in the central nervous system. The antioxidant and anti-apoptotic properties of PRDX5 recombinant protein have also been demonstrated. The pathophysiology of brain lesions associated with cerebral palsy is probably multifactorial and likely involves, among other factors, excess release of glutamate, inducing the so-called excitotoxic cascade, and excess production of free radicals. Antioxidants would therefore limit the severity of these brain lesions. Peroxiredoxins (PRDXs) are a family of naturally-occurring antioxidant enzymes. PRDX5 is a recently cloned member of this expanding family. It displays peroxidase activity and possesses targeting sequences for peroxisomes and mitochondria. The neuroprotective effects of PRDX5 against neonatal excitotoxic challenge have been studied in vivo and in vitro.

In vivo excitotoxic stress was produced by intraneopallial injection of ibotenate (acting on NMDA and metabotropic receptors) or s-bromowillardiine (acting on AMPA-kainate receptors) to postnatal day (P) 5 mouse pups. The pups were killed 5 days later and brains were processed for histology to determine the size of neocortical plate (mimicking lesions of full-term human infants) and periventricular cystic white matter lesions (mimicking PVL). Recombinant PRDX5 (0.1-200 mg/kg) was administered IP immediately after the excitotoxin injection. Effects of PRDX5 were compared to those obtained with reference antioxidants including N-acetylcysteine (0.25-250mg/kg) or catalase-polyethyleneglycol (catalase-PEG; 6,000-600,000 U/kg). Controls received IP saline. In vitro, neocortical neurons were exposed to 300microM NMDA in the absence or presence of 0.1-100 microM PRDX5 alone or 10 microM PRDX5 + 10 microM dithiothreitol (DTT, a classical electron donor). Apototic nuclei were counted following chromatin staining with Hoechst 33258. Systemically administered PRDX5 induced a dose-dependent neuroprotection of the ibotenate-induced, but not s-bromowillardiine-induced, lesions of both the cortical plate and the white matter (maximum reduction of 63% of the lesion size). N-acetylcysteine and catalase-PEG mimicked PRDX5 effects on excitotoxic lesions. In vitro, PRDX5 and DTT displayed a synergetic neuroprotective effect on NMDA-induced neuronal death.

This example shows that free radical production participates to the formation of NMDA receptor-mediated brain lesions in newborn mice and that antioxidant drugs such as PRDX5 are

neuroprotective in these models. Thus, the use of SEQ ID NO:1 and the polypeptide encoded by the polynucleotide provide similar treatment possibilities. Applicants assert that the potential for the presently claimed vector and transformed cell as both diagnostic tools and in treatment of lung injuries and diseases or disorders related to oxidative stress would not have been obvious to one of skill in the art. Therefore, Applicants respectfully request withdrawal of the rejection on this basis.

Conclusion

Should any issues arise which may delay prosecution of the present application the Examiner is respectfully invited to contact the under-signed attorney at the telephone number below.

Please charge any additional fees, including any fees for additional extension of time, or credit overpayment to Deposit Account No. 11-1410.

Respectfully submitted,

KNOBBE, MARTENS, OLSON & BEAR, LLP

Dated: 2/6/02

By: _____

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VERSION WITH MARKINGS TO SHOW CHANGES MADE

Deletions are in **[bold and brackets]** and insertions are underlined.

IN THE ABSTRACT:

Please insert the Abstract attached hereto, following the VERSION WITH MARKINGS TO SHOW CHANGES MADE, as page 24 of the application as filed.

IN THE CLAIMS:

Please cancel Claims 6, 7, and 8.

Please amend the following claim:

5. (THREE TIMES AMENDED) An isolated or purified polynucleotide **[encoding the amino acid sequence according to claim 1 and more than 70% homologous to]**comprising SEQ ID NO:1 or its complementary strand.

12. (THREE TIMES AMENDED) A diagnostic device comprising the nucleotide sequence of claim 5**[or a portion thereof]**.

14. (THREE TIMES AMENDED) A pharmaceutical composition comprising a pharmaceutically acceptable carrier and the nucleotide sequence of claim 5**[or a portion thereof]**.